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Dietary Conjugated Linoleic Acids (CLA) and Body Fat Changes

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Conjugated linoleic acid consumption by mice reduced body fat. A parallel effect is implied for humans that consume beef.

Summary

A mixture of CLA isomers was fed to mice at 0, 1, and 2% of the diet for 5, 12, or 14 days. Dietary CLA caused a reduction of body fat approaching 50% but did not cause a loss of total body weight. Mice fed CLA also experienced programmed cell death (apoptosis) of fat cells. Certain (not all) CLA isomers are natural components of beef and dairy products but not other foods. Therefore, these observations prompt speculation of an additional health benefit, reduced adiposity — to humans that eat ruminant-derived foods.

Introduction

Conjugated linoleic acids (CLA) are produced by anaerobic ruminal metabolism and subsequent animal metabolism. Consequently, ruminant animal fats contain substantial CLA (~.5%) and are by far the predominant source of CLA in the human diet. Initial investigations revealed cancer-preventing effects of CLA. Others have reported that feeding CLA to mice for 6 weeks can increase energy expenditure and reduce body fat. The mechanism by which CLA mediate these effects is uncertain, but there is evidence that CLA influence expression of fat metabolizing genes. The objective of our research was to determine effects of feeding CLA to mice on energy expenditure, feed intake, body fat and apoptosis in white adipocytes. The rationale is that the response of mice to dietary CLA may predict the response of humans to dietary beef or dairy fats.

Procedure

Diets

Conjugated linoleic acid was mixed into a purified base diet. Soy oil was replaced (1:1) with CLA to create diets of 0, 1 and 2% CLA. This CLA is a mixture of isomers with approximately 44% being the type (c9/t11) that predominates in ruminant fats, and 41% is t10/c12.

Experiment 1

Ninety 10- to 12-wk-old male mice were housed individually at 22° C and randomly assigned to one of the three experimental diets. Feed intake and body weight were measured daily. Direct calorimetry was used to measure heat loss during a four-hour period beginning at 1700 on day 9 for each replicate. On the day of calorimetry, feed was unavailable from 1200 until 1900. Thus, heat loss was determined in the fasted and in the refed state for each animal. Heat loss was determined at one-minute intervals and collected every 30 minutes for two hours in each state. Water was not available in the calorimeter chambers. Three days after calorimetry, between 0800 and 1000, mice were sacrificed by CO_2 asphyxia. Brown, epididymal and retroperitoneal fat pads and livers were removed and weighed. Twenty-one retroperitoneal fat pads were analyzed for apoptosis (programmed cell death) by a DNA laddering assay.

Experiment 2

Twenty obese M16 strain retired male breeders were randomly assigned to one of three CLA diets: 1) 0% for 12 days; 2) 2% for 14 days; and 3) 0% for 9 days followed by 2% for 5 days. Body fat, body weight, and apoptosis were assayed as in experiment 1.

Results

Experiment 1

Feed intake (g/day) by mice fed 0, 1, and 2% CLA for 12 days was 5.0, 4.7, and 4.4 (SE = .11; $P < .05$) respectively. Despite consuming less feed, the mice fed 1 and 2% CLA expended as much energy as the controls. After consuming CLA for 12 days, mice had considerably less body fat than contemporaries which were not fed CLA (Figure 1). The fat cells from mice fed 2% CLA presented more apoptosis than cells from control mice ($P < .10$).

Experiment 2

Consumption of 2% CLA diet for either five or 12 days caused a significant loss of body fat in all of the depots measured (Figure 2). In spite of this

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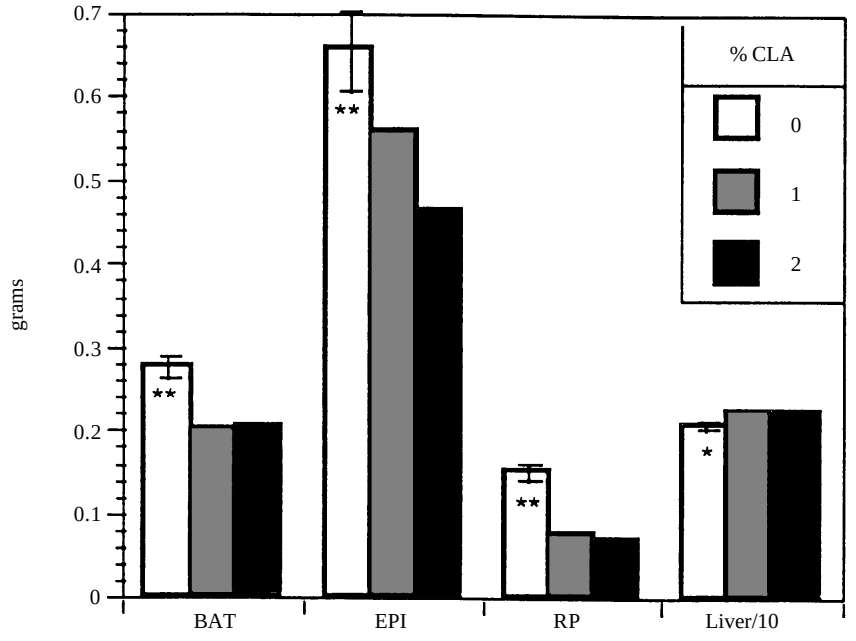

Figure 1 Experiment 1 Weight of Brown (BAT), Epididymal (EPI), and Retroperitoneal (RP) Fat Pads, and of Liver (scaled to 10% of actual weight). **CLA effect (P<.01). *CLA effect (P<.10). Error bars represent SEM.

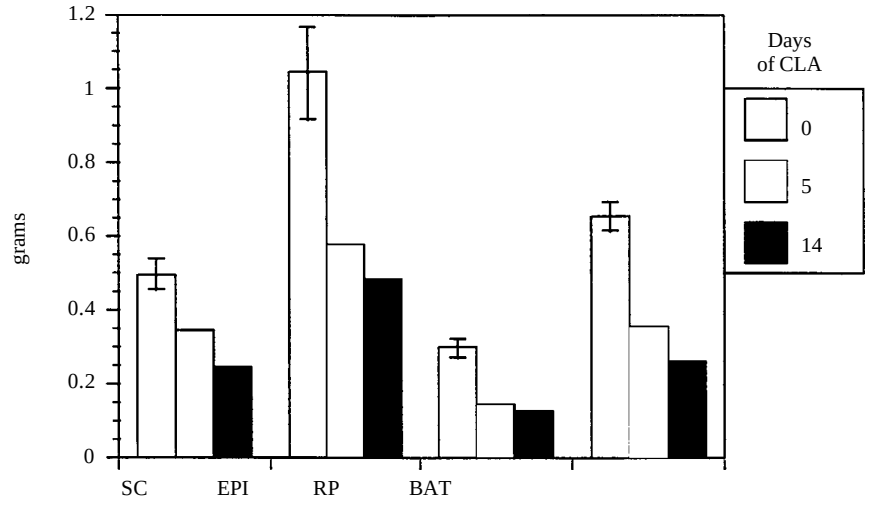

Figure 2. Experiment 2 Weight of Subcutaneous (SC), Epididymal (EPI), Retroperitoneal (RP), and Brown (BAT), Fat Pads. **CLA effect (P<.01). Error bars represent SEM.

loss of body fat, total body weight increased in animals fed CLA versus control ($P < .05$). Consistent with the results of experiment 1, analysis of retroperitoneal fat pads indicated that dietary CLA caused programmed cell death in fat cells ($P < .03$).

In conclusion, feeding mixed isomers of conjugated linoleic acid to mice causes a rapid (within 5 d) loss of body fat, no loss of total body weight, and appears to cause apoptosis of fat cells. Perhaps this apoptosis mediates the specific loss of body fat caused by CLA consumption.

It is inviting to hypothesize that ruminant animal-derived human foods can provide an anti-obesity benefit. This conclusion cannot be drawn with much certainty at the moment. Although ruminant fats are the only significant source of CLA in the human diet, most of the CLA in ruminant fat is c9/t11. Our experimental CLA mixture contained nearly as much t10/c12 as it did c9/t11. Therefore, the effect we observed in mice could be due to an isomer of CLA which is not predominant in any food. Furthermore, there is a question about whether the amount of CLA necessary to cause a benefit could be supplied by a relatively normal diet. Certainly, the amount of CLA which we fed mice (1% and 2%) couldn't be obtained naturally given that beef and milkfat are .5% CLA. However, some research conducted at the University of Georgia indicates that .25% CLA causes a loss of body fat in rats. We have not tested such low concentrations.

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